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RESEARCH AREA

Our research group is interested in understanding the mediobiological significance of the efferocytosis (clearance of dead cells) program. Every day 1 billion cells die in our body, mainly as part of the tissue cell turnover. But cells are dying also during infections, or tissue injury. Dead cells generated as part of the tissue turnover are cleared by tissue resident macrophages, while those generated during ipathological processes by bone marrow-derived macrophages. These cells not only engulf and degrade dead cells, but during efferocytosis an anti-inflammatory, tissue regenerating program is activated in them. If this program is disturbed, chronic inflammatory diseases and wound healing deficiencies develop.We are investigating efferocytosis in the thymus, where immature thymocytes develop, in skeletal muscle injury and the obese adipose tissue by using various genetically modified mice. Our aim is to treat chronic inflammatory diseases and wound healing more efficiently by understanding the elements of the efferocytosis program.

TECHNIQUES AVAILABLE IN THE LAB

- Planning and maintaining experiments with laboratory mice.
- Experiments with cell cultures.
- Isolation of primary cells from animals
- FACS analysis of cells
- Histological stainings and analysis of tissues
- Western blot technique
- qRT PCR technique
- ELISA
- Insulin and glucose tolerance tests
- Efferocytosis assay

SELECTED PUBLICATIONS

Fige, É., Sarang, Z., Sós, L., **Szondy, Z.** (2022) Retinoids promote mouse bone marrow-derived macrophage differentiation and efferocytosis via upregulating bone morphogenetic protein-2 and Smad3. **Cells 11(18):** 2928.

Sós, L., Garabuczi, É., Sághy, T., Mocsár, G., **Szondy, Z.** (2022) Palmitate inhibits mouse macrophage efferocytosis by activating an mTORC1-regulated rho kinase 1 pathway: therapeutic implications for the treatment of obesity. **Cells 11(21):** 3502.

Garabuczi, É., Tarban, N., Fige, É., Patsalos, A., Halász, L., Szendi-Szatmári, T., Sarang, Z., Király, R., **Szondy, Z.** (2023) Nur77 and PPARγ regulate transcription and polarization in distinct subsets of M2-like reparative macrophages during regenerative inflammation. **Front Immunol 14:** 1139204.

Gruper, Y., Wolff, A. S., Glanz, L., Spoutil, F., Marthinussen, M. C., Osickova, A., Dobes, J., Kadouri, N., Ben-Nun, O., Binyamin, A., Lavi, B., Givony, T., Khalaila, R., Gome, T., Wald, T., Mrazkova, B., Sochen, C., Marthinussen, M. C., Besnard, M., Ben-Dor, S., Feldmesser, E., Orlova, E. M., Felszeghy, S., Papp, T., Hegedűs, C., Lampé, Z., Sedlacek, R., Davidovich, E., Tal, N., Shouval, D. R., Shamir, R., Guillonneau, C., **Szondy, Z.**, Lundin, K. E. A., Osicka, R., Prochazka, J., Husebye, E. S., Abramson, J. (2023) Autoimmune amelogenesis imperfecta in patients with APS-1 and coeliac disease. **Nature 624(7992):** 653-662.

Tarban, N., Papp, A. B., Deák, D., Szentesi, P., Halász, H., Patsalos, A., Csernoch, L., Sarang, Z., **Szondy, Z.** (2023) Loss of adenosine A3 receptors accelerates skeletal muscle regeneration in mice following cardiotoxin-induced injury. **Cell Death Dis 14(10):** 706.